

The undesirable symptoms which are sometimes noticed during the application of electricity may not be injurious, however uncomfortable they are for the time—they do not, Dr. Lincoln thinks, absolutely indicate that the treatment is not doing good.

SALICYLATE OF SODA.—MM. Bochefontaine and Chabert have experimented upon the physiological action of salicylate of soda, and recently reported their results to the Acad. des Sciences, Paris. According to these investigators, it is neither a cardiac nor a muscular poison, nor does it act on the extremities of the nerves. Its action appears to be limited to the gray substance.

ACTION OF PILOCARPINE AND ATROPINE ON THE SWEAT GLANDS.—Dr. B. Luchsinger (Pflüger's Archiv., Bd. 15, S. 482,) has continued his researches on the mechanism of perspiration in the cat. Pilocarpine, the alkaloid of jaborandi injected subcutaneously in the dose of 0.01 grm. (1.7 grain), causes violent perspiration of the paws. That this is in part due to stimulation of the nerve-terminations is proven by the persistence of sweating after section of the sciatic nerve. If, however, a sufficient time has elapsed since the section to permit complete degeneration of the nerve, the corresponding sweat glands are no longer excited to action by pilocarpine. But the alkaloid stimulates also the origin of the sweat nerves in the cord, since on cutting off the blood supply to the hind legs and severing the cord from the medulla, pilocarpine will still induce perspiration.

The effects of the above mentioned dose are checked by 0.003 grm. atropine. Even electric stimulation of the sciatic nerve fails now to excite the sweat glands. But if, now, another dose of 0.01 grm. pilocarpine is injected under the skin of one paw, the secretion returns at that spot and irritation of the nerve is again followed by perspiration, while the paralytic effects of atropine are still manifest in the glands of the other foot. These well executed experiments prove a perfect reciprocal antagonism between pilocarpine and atropine in the action on the sweat glands. H. G.

NEUROTOMY.—At the International Medical Congress at Geneva (Sept. 9-15, 1877, rep. in *Gaz. des Hôpitaux*), M. Letievent defended the operation of neurotomy in rebellious cases of neuralgia. It was, he said, by no means as unreliable a means as had been stated, and no more dangerous than any other insignificant wound. Care should be taken not to cut the nerve until it was fairly laid open to view, and that no vessels, etc., are included in the section, to avoid hemorrhage. Care should also be taken to not tear or roughly pull the nerve so as to shock its encephalic roots.

M. Letievent has performed twenty-two neurotomies in sixteen cases and twelve individuals. In all the cases it was performed on account of intense and persistent pains, resisting all other treatment. The duration of

these neuralgias was twenty years, twelve years, five years, several years, and many months. In ten of the twelve persons a cure was obtained. There were two cases of rapid relapse, in these the method was not persistently tried. The cures had been watched and followed for the periods of five years, three years and a half, and eighteen months. In two cases only the immediate effects could be observed.

M. Letievant concluded with the expression of his opinion that neurotomy admitted in theory, has by its success forced itself to be admitted in practice.

CAMPHOR—C. Wiedeman, *Arch. f. exp. Path.* VI., 216, (abstr. in *Centralblatt*, No. 37.) The well known epileptiform attacks which follow camphor poisoning in mammals, do not occur in rabbits if the cervical cord is separated from the medulla, and must, therefore, have their start from above this point of section, and not be due to the spinal cord. In winter frogs no convulsions appear. In summer frogs a convulsive extension of the posterior extremities may be produced by mechanical irritation of the head or back shortly before the appearance of the camphor paralysis. (Harnack and Witkowsky have also observed fibrillary contractions in frogs from the administration of camphor.) The reflexes exist with increased intensity for a while after voluntary movements have become very much weakened, but delayed; they are lost first in the stage of general paralysis. Electric irritation of the medulla or the cord then has little or no effect, faradic excitation of the sciatic is also of little effect, while, on the contrary, direct excitation of the muscles produces powerful contractions. If one limb exclusive of the nerve was separated from the body before the poisoning, then this nerve remained normally irritable. But contractions of this limb can still be excited from the cord. (The author has not undertaken the more suitable experiment for the testing of this question, by exclusion of the blood supply). He is of the opinion that there is a curare-like action of the camphor on the terminations of the motor nerves and a paralysis of the longitudinal and (*reflex*) cross-conduction of the cord, which he considers to be the causes of the almost absolute absence of convulsions in the frogs. The stoppage of the heart (in frogs) by muscarine is counteracted or hindered by camphor (Harnack). Irritation of the vagus also produced no stoppage of the heart. As Harnack and Witkowsky found with physostigmin, which has a similar action on the heart, so also with camphor, after the introduction of substances paralyzant to the muscles (tartrate of copper and soda), stoppage of the heart was again produced by irritation of the vagus and by muscarine, which H. and W. took as a proof that such substances as physostigmin excite the cardiac muscle, and that the inhibitory influence of the vagus is too weak to contend against this super-excitation, but when the muscle is weakened by the poison, excitation of the vagus by electricity, or muscarine again obtains the upper hand, and again the inhibition of the heart's action takes place. Also in curarized cats in which artificial respiration was kept up, there was seen under the action of cam-